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Demographic and clinical characteristics reflect different phenotypes of osteoarthritis in the lumbar spine: the Johnston County Osteoarthritis Project

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Abstract

Objective: Determine if associations between demographic, appendicular joint osteoarthritis (OA), clinical characteristics reflect different phenotypes of OA in the lumbar spine.

Methods: Participants were from the Johnston County OA Project. Demographics consisted of age, sex, and race (White and African American [AA]) and clinical characteristics consisted of body mass index (BMI), low back pain and injury, and knee, hip and hand osteoarthritis (OA).

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All authors disclose they have no financial or personal relationships with other people or organizations that could potentially and inappropriately bias their work and conclusions.

Participants were categorized as spine OA (SOA), facet joint OA (FOA), both SOA and FOA, or neither SOA nor FOA (referent group). Multinomial regression models were used to determine odds ratios (OR) and 95% confidence intervals (CI).

Results: The average age (n=1,793) was 66.2 years (SD=10.1) and BMI was 30.7 (SD=6.2). A majority (63.8%) were women (n=1,144) and 31.8% (n=570) AA. Neither SOA nor FOA was present in 18.0%; 22.8% had FOA, 13.2% had SOA and 46.0% had both SOA and FOA. In adjusted analyses, AA were less likely to have FOA (OR=0.68 95% CI 0.49, 0.95) and both SOA and FOA (OR=0.51 95% CI 0.37, 0.70). Women were more likely to have FOA (OR=1.71 95% CI 1.24, 2.36). BMI ≥30 kg/m^2 was associated with FOA (OR=1.76 95% CI 1.28, 2.42) and both SOA and FOA (OR=1.85 95% CI 1.37, 2.51). Knee OA was associated with all three groups while lower back injury was associated with only SOA. Participants with hip OA were less likely to have FOA.

Conclusion: Race, sex, BMI, hip OA, and lower back injury may inform different OA phenotypes in the lumbar spine.

Chronic low back pain (cLBP) impacts over 31 million Americans at any given time (1), has increased threefold in prevalence over a 10-year period (2), and results in $100-$200 billion per year in total U.S expenditures (3). Chronic LBP can be due to a number of etiologies, including degeneration of the intervertebral disc (IVD) and facet joint osteoarthritis (FOA). Improved diagnosis of these poorly understood etiologies is critically important. (4–10) While most treatment options for cLBP have minimal side effects or harms, some complications do exist (11–13). A better understanding of the etiological process of spine degeneration may improve the delivery of interventions to specific lumbar spine structures.

Osteoarthritis (OA) in the spine has been characterized by the combination of mild radiographic disc space narrowing (DSN); analogous to joint space narrowing in an appendicular joint) and at least mild radiographic vertebral osteophyte formation (at the same lumbar level) (14, 15) based on a radiographic atlas definition (i.e., Kellgren-Lawrence)(16). This type of definition for OA in the spine is commonly referred to as spondylosis. However, the pathophysiology of IVD degeneration in the spine as an OA process is controversial (17). This is in part due to anatomical differences that exist within the IVD that do not necessarily exhibit the same pathophysiology as OA in the knee, hip or hand (7). Some consider OA in the lumbar spine to be confined to the facet or zygapophyseal joint, since this is the only structure in the spine that is classified as a synovial joint (i.e., contains articular cartilage, synovial lining and a joint capsule) (18). We have identified differences in biochemical biomarker profiles (19) and appendicular joint OA (i.e, knee, hip, and hand OA)(7) between IVD degeneration and FOA that suggest a different pathophysiologic processes may exist between these two individual radiographic features.

The finding of biochemical differences underlying different phenotypes of OA in the lumbar spine leads to the question of whether demographic, appendicular joint OA, and clinical characteristics also differ by phenotype. Identifying clinical phenotypes (i.e., well-defined and mutually exclusive OA sub-types(20)), defined by pathology of different lumbar structures may lead to better and more precise intervention approaches for preventing, diagnosing, or treating OA of the lumbar spine. Identifying, differences in demographic,

appendicular joint OA, and clinical characteristics across two definitions of OA in the lumbar spine may facilitate the identification of distinct phenotypes. Therefore, the purposes of this study were to: 1) describe the demographics, appendicular joint OA, and clinical characteristics across lumbar spine and facet joint OA and 2) determine if lumbar spine OA phenotypes are identified by demographics, appendicular joint OA and clinical characteristics. We hypothesized that phenotypes could be identified by distinct demographics, appendicular joint OA, and clinical characteristics.

Methods and Materials

Participants

Details of the sampling strategy and recruitment methods used for the Johnston County Osteoarthritis Project (JoCo OA) are described elsewhere (7, 21). The primary purpose of the JoCo OA is to determine the incidence, prevalence and progression of OA. This ongoing, longitudinal study of OA includes African American (nearly 30% of the cohort) and White participants living in a largely rural county in North Carolina. Civilian, non-institutionalized residents aged 45+ years from six townships in Johnston County were enrolled between 1991 and 1998 (n=3187, Original Cohort), and additional residents were enrolled from 2003–2004 (n=1015, Enrichment Cohort). Participants in JoCo OA completed follow-up clinic and interview data collection approximately every 5 years. Lumbar spine radiographs were added to JoCo OA in 2003. Since spine radiographs were added after the first follow-up for the Original Cohort participants had concluded, our cross-sectional data come from (a) participants who received their first clinic visit at T1 (2003–2004; n=1,055 including 40 participants who had enrolled in the study at T0 but never completed a clinic visit) and (b) the Original Cohort participants returning for their second follow-up (2006–2010; n=1,088) (Figure 1). Since the Enrichment Cohort aimed to supplement the sample for AAs and younger participants, participants enrolled during 2003–2004 were younger (mean age 59.3 vs. 65.8 years) and more likely to be AAs (40% vs. 28%) than Original Cohort participants at first follow-up (1999–2003); the two groups did not differ according to sex (22). Since JoCo OA is a community-based observational study, we followed the Strengthening The Reporting of Observational Studies in Epidemiology (STROBE) statement (23), a 22-item checklist that was developed to ensure accurate and complete reporting of observational studies.

Demographics and Clinical Characteristics

Demographic data were collected by clinical interview and examination, including age and body mass index (BMI) at the time of interview (calculated from height measured without shoes and weight measured with a balance beam scale), race (White / AA), and sex. Low back symptoms were collected at clinical interview by asking participants to answer “yes” or “no” to the question “On MOST days do you have pain, aching or stiffness in your lower back”?

Radiographic Spine Evaluation

By protocol, women of reproductive age (<50 years of age) were excluded from having lumbar spine radiographs. Lateral lumbar spine films were taken with the participant lying

on his/her left side with the central beam centered at the lumbar spine. The Burnett Atlas(24) was used to grade lumbar spine radiographic features of FOA, DSN and osteophytes (OST). FOA was graded as absent or present at each lumbar level while DSN and OST were graded in a semi-quantitative fashion (0=none, 1=mild, 2=moderate and 3=severe). OST was assigned for each superior and inferior aspect of the anterior face of the lumbar vertebra. All lateral lumbar spine radiographs were graded at each lumbar level by an experienced single bone and joint radiologist who has been the radiologist for the JoCo OA Project since its inception. The intra-rater reliability of this radiologist have been reported previously with a weighted kappa (wK) for FOA wK=0.73, for DSN wK=0.89, and for OST wK=0.90 (19).

Lumbar Spine OA Phenotypes

Spine OA (SOA) was defined by the presence of at least a mild OST (either superior or inferior of the anterior face of the vertebrae) and mild DSN at the same level of the lumbar spine for any level of the lumbar spine. FOA was categorized as present and absent at any level of the lumbar spine. From these two different coding schemes, we developed our spine degeneration phenotypes. Participants were categorized as having no FOA or SOA, FOA only, SOA only or a combination of both FOA and SOA.

Knee, Hip and Hand Osteoarthritis

Participants completed weight bearing postero-anterior knee radiography of both knees with a Synaflexer™ (CCBR-Synarc, San Francisco, CA) positioning device and bilateral hip radiography with supine anteroposterior pelvis radiographs. The primary reason for a participant not having knee radiographs was presence of knee arthroplasty. The primary reason for missing hip radiographs was exclusion of this procedure for women of reproductive age (<50 years). Postero-anterior hand radiographs were obtained with the beam focused on the third metacarpophalangeal joint; hand radiographs were graded for 30 hand joints bilaterally (the distal interphalangeal [DIP], proximal interphalangeal [PIP], metacarpophalangeal [MCP], carpometacarpal [CMC] and thumb IP and MCP joints). All knee, hip, and hand radiographs were read for Kellgren-Lawrence (K-L)(25) score by a single bone and joint radiologist. Inter-rater and intra-rater reliability have been reported previously with a wK of 0.86 and 0.89 for both the hip and knee (26). Hip and knee OA, for these analyses, were defined as a K-L score of 2–4 in at least one extremity. Hand OA was defined, similar to a previous definition, as at least one extremity with a K-L grade of 2–4 in one DIP and in at least 2 other interphalangeal joints or CMC joints affected across both hands (15).

Statistical Analysis

Descriptive statistics were generated for the total sample and each potential phenotype in the form of means and standard deviations or median and interquartile ranges for continuous covariates and counts and proportions for categorical covariates. Analysis of variance was used for continuous variables, and chi square tests were used for categorical variables to determine differences across FOA only, SOA only or the combination of both FOA and SOA.

We used multinomial regression to estimate unadjusted and adjusted associations between demographic, clinical characteristics, and peripheral joint OA with potential OA in the lumbar spine phenotypes managed as a nominal response variable. This regression technique compares each of the response categories to the common referent group. Odds ratios with corresponding 95% confidence intervals were the measure of association. All analyses were conducted in SAS 9.4 (SAS Institute Inc., Cary, NC), and statistical significance was set at $p<0.05$.

Results

Figure 1 illustrates the selection of participants for inclusion in the analyses. Participants who attended study visits during 2003–2004 (n=1055) or 2006–2010 (n=1088) were included. A higher number of missing lumbar spine radiographs (n=203) were present at the T1 time frame compared to T2 (n=20). This was primarily due to the higher number of women of childbearing age that were excluded by protocol. Of those, a small number (n=127) were missing covariate data leaving 1,793 with lumbar spine radiographs and complete covariate data.

Table 1 describes the demographic and clinical characteristics for the included participants. Of the 1,793 participants with complete lumbar spine radiographs the average age was 66.2 years (SD=10.1) and BMI was 30.7 (SD=6.2). A majority n=1,144 (63.8%) of participants were women and 31.8% (n=570) were African American. A substantial proportion of participants reported the presence of low back pain (39.7%) and of those with pain it was moderate in 17.6% and severe in 9.1% of participants. Self-reported back injury was rare (2.3%) amongst participants. Peripheral joint OA was present in the knee for 39.4%, in the hip for 32.8% and in the hand for 30.6% of participants. The distribution of OA in the spine consisted of 18.0% having neither SOA nor FOA, 22.8% having FOA only, 13.2% having SOA only and 46.0% having both SOA and FOA.

Table 2 describes the groups of OA in the lumbar spine across demographic and clinical characteristics. Participants in the neither SOA nor FOA were significantly ($p<0.0001$) younger (59.6, SD=9.1) and had a significantly ($p=0.0002$) lower BMI (29.5 kg/m^2, SD=5.8) compared to the other potential SOA phenotype groups. A significantly ($p<0.0001$) greater proportion of men and African Americans were in the neither SOA nor FOA group. The distribution of knee OA presence was significantly ($p<0.0001$) lower in the neither SOA nor FOA group whereas the distribution was greater in the both spine and FOA group. A significantly ($p=0.0002$) greater proportion (53.4%) of participants with hip OA was found in the both SOA and FOA group. Similarly, a significantly ($p<0.0001$) greater proportion (60.5%) of hand OA was present in the both SOA and FOA group. No significant differences were found across any of the groups with the presence or severity of low back pain.

Table 3 describes the unadjusted and adjusted associations between each demographic and clinical characteristics across each category of OA of the spine relative to the absence of both SOA and FOA group. After adjustment for all other demographic and clinical characteristics strong associations continued across categories of age with the strongest associations with the both FOA and SOA group. Strong significant and similar associations

were found between BMI ≥30 and the FOA only group (OR=1.76 ((95% CI 1.28, 2.42) and the both FOA and SOA group (OR=1.85 ((95% CI 1.37, 2.51)). African Americans were 32% less likely to have FOA only (OR=0.68 (95% CI 0.49, 0.95)) and 49% less likely to have both SOA and FOA (OR=0.51 ((95% CI 0.37, 0.70)). Women had 71% higher odds of FOA only (OR=1.71 ((95% CI 1.24, 2.36)) compared to men. Participants reporting a previous back injury had a strong significant association with SOA only (OR=3.11 ((95% CI 1.09, 8.91)). The only significant association with low back symptoms was found with the both FOA and SOA group (1.35 (95% CI 1.00, 1.84). Similar associations were found between radiographic knee OA and FOA only (OR=1.78 ((95% CI 1.23, 2.57)) and SOA only (OR=1.79 ((95% CI 1.19, 2.70)). The association between radiographic knee OA and the both SOA and FOA group was substantially stronger (OR=2.50 ((95% CI 1.78, 3.52)). There was 31% lower odds of hip OA among the FOA only group (OR=0.69 95% CI 0.48, 0.98). No significant association was found between low back pain severity or hand OA for any of the groups.

Discussion

To our knowledge, this is the first study to determine if demographic and clinical characteristics have the potential to be used to develop phenotypes for lumbar spine and facet joint OA. We identified a substantial proportion of participants that had radiographic evidence of OA isolated to the lumbar spine or the facet joint. Some similarities and several variations in the associations between demographic and clinical characteristics in adjusted models support our hypothesis that groups differing by these radiographic manifestations may belong to separate clinical phenotypes.

Based on previous biochemical biomarker analyses in JoCo OA, we identified a subset (n=555) of participants with radiographic FOA but without radiographic DSN. (19) Since much of the previous literature has focused on a direct relationship between DSN leading to FOA, this large subset suggests a unique finding, (27) supporting a suspicion that in in a subgroup of ~20%, FOA may precede IVD degeneration (28). Furthermore, our previous work suggests the potential independence of FOA and SOA in certain individuals (7, 19). Also, in the present study, a subgroup was identified without any radiographic SOA nor FOA. Considering our analyses, on average, involve older adults with a traditionally higher prevalence of IVD degeneration and FOA, the finding of a group of participants without any radiographic evidence of either entity suggests a potential SOA resistant phenotype. As suggested by some and supported by this work, similarities may exist in the pathophysiology between appendicular joint OA and degeneration of the spine (29). However, our work further suggests that traditionally defined OA, like that found in the facet joint of the lumbar spine (i.e., contains articular cartilage, synovial lining and a joint capsule), may have a different etiological process than joint space defined OA (i.e., disc space narrowing and osteophyte formation).

Interestingly, we identified a strong statistically significant association of self-reported history of low back injury only in the SOA group. Although joint injury has certainly been linked to OA of the knee, (30, 31) to our knowledge, no other studies have reported an association between history of lower back injury and SOA. Participants reporting injury to

the lower back may have suffered a bulge of the IVD, which can be prevalent but asymptomatic in the general population. (1) The influence of disc bulge on mechanical composition and properties of the disc may lead to accelerated IVD degeneration (32, 33). However, the exact mechanisms by which injury may affect the composition and mechanical properties of the lumbar spine leading to IVD degeneration cannot be addressed with the imaging used for this study (i.e., plain film radiographs). In addition, our sample size of participants that reported previous back injury was small and resulted in an imprecise confidence interval. This result should be examined with more precise imaging (i.e., MRI) and in a longitudinal study to determine if a history of low back injury is a risk factor for incident and/or worsening IVD degeneration.

We identified a significant association between LBP and the FOA and SOA group but not the FOA only or SOA only group. However, the strength of association found with the both FOA and SOA group was similar to the SOA group suggesting that DSN may be the primary contributor to this similarity. Although this has been a topic of discussion in the literature, a meta-analysis and studies by our group have identified consistent and significant associations between the individual radiographic feature of DSN and LBP (4, 7, 17). In our previous work (7), as well as the work of others (8, 34), the individual radiographic feature of DSN alone without the presence of osteophytes has been commonly used as an indicator of spine degeneration. The consistent weak associations between vertebral osteophytes and LBP that have been reported in meta-analyses may have attenuated the associations in our current study. For understanding etiologies of LBP, these findings reinforce the importance of examining individual radiographic features rather than composite definitions of OA in the lumbar spine.

Our study found that several demographic, clinical characteristic and peripheral joint OA variables are significantly associated with FOA only and with the combination of FOA and SOA, whereas no significant associations were found with the SOA only group. Although their study did not examine different SOA groups, similar to Kalichman et al (35) we identified a strong association between BMI and FOA but not between BMI and SOA only. Although, BMI has been identified as a strong risk factor for knee (36), hip (37) and hand OA (38), our previous work with this cohort has demonstrated no association between BMI and DSN (7). In fact, the only demographic factor that was significantly and strongly associated with SOA (DSN and not FOA) was increasing age. Similar to our previous work (7), AA’s were far less likely have FOA with similar strength in association to the both FOA and SOA group suggesting FOA is the primary contributing structure to this association. Our previous work has found a significant association between FOA and knee OA (7). However, in that study FOA was not isolated and included individuals with DSN. The strong associations found in the present study with knee OA and all three lumbar SOA groups suggests some participants may have multisite OA, which may be more genetic in nature. We have also reported in previous work (7) that hip OA does not seem to follow the same etiological process as spine degeneration. Our current findings further support this work in that those with hip OA are far less likely to have the FOA. These findings strengthen the argument that the etiology of spine degeneration may be influenced by different demographic and clinical characteristics, some that are similar to peripheral joint OA and some that are distinct.

There are several strengths to our study including a well-defined population, large sample size and protocol driven approach to data collection. However, our study is not without some limitations. The primary limitation of this study is its cross-sectional design; thus, we could not determine the temporality between demographic and clinical characteristics and the onset of spine degeneration. Lateral lumbar spine radiographs may not be the optimal image or view for FOA, which could lead to non-differential misclassification of FOA status. However, prevalence estimates of FOA based on lateral spine radiography (7) are similar to those previously reported based on computed tomography scans (39). Per protocol, we excluded women of childbearing age; these factors may limit generalizability. We combined two unique time points of JoCo OA with known differences in age, sex and race between these time points, which may influence our estimates given the differences in associations between sex and race with OA of the lumbar spine found in these analyses. However, our intent was to capture the first lumbar spine radiograph for a participant in the cohort.

In summary, OA in the lumbar spine is a unique process that differs between degeneration of the IVD along with osteophyte formation and the OA process found in the facet joint. Collectively these findings suggest the pathophysiologic processes may vary across different OA phenotypes in the lumbar spine. Increasing associations across categories of age suggest changes over time, however longitudinal studies may elucidate whether those with FOA only or SOA only remain an isolated lumbar SOA phenotype over time. In these analyses, race, sex, BMI, hip OA, and history of back injury may inform different OA phenotypes in the lumbar spine.

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Highlights

- Four subgroups of individuals with osteoarthritis (OA) of the lumbar spine were identified, consisting of those without any spine OA (ie., intervertebral disc degeneration and vertebral osteophytes) and facet joint OA, those with facet joint OA only, those with spine OA only and those with both spine OA and facet joint OA.
- African Americans and females were significantly less likely to have facet joint OA only.
- Those with spine OA only were more likely to have a history of low back injury while those with low back pain were more likely to have both spine OA and facet joint OA.
- Knee OA was associated with all three groups of OA in the lumbar spine while those with hip OA were less likely to have facet joint OA.
- The differences in demographic and clinical characteristics found in these analyses may inform different OA phenotypes of the lumbar spine.

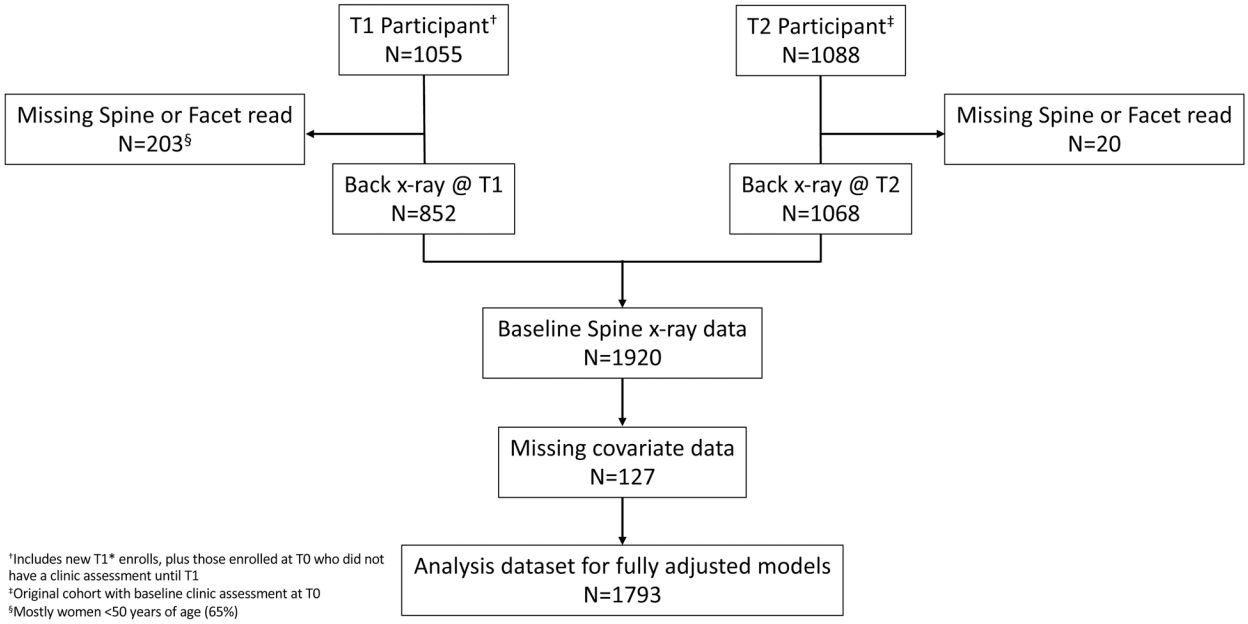

Figure 1.
Participants with lumbar spine radiographs in the Johnston County Osteoarthritis Project.

Table 1.

Characteristics of participants with complete lumbar spine radiographs (n=1793)[§]

Baseline Characteristic	Level	Value
Age	Age, Years, Mean (SD)	66.2 (10.1)
	Age <55, n (%)	268 (14.9)
	Age ≥55-<65, n (%)	601 (33.5)
	Age ≥65-<75, n (%)	553 (30.8)
	Age ≥75, n (%)	371 (20.7)
BMI	Mean BMI, Mean (SD)	30.7 (6.2)
	BMI <25, n (%)	303 (16.9)
	BMI 25-<30, n (%)	616 (34.4)
	BMI ≥30, n (%)	874 (48.7)
Gender	Men, n (%)	649 (36.2)
	Women, n (%)	1144 (63.8)
Race	African American, n (%)	570 (31.8)
	White, n (%)	1223 (68.2)
Back Pain	No Back Pain, n (%)	1082 (60.3)
	Has Back Pain, n (%)	711 (39.7)
Pain Severity	No Back Pain, n (%)	1082 (60.3)
	Mild Back Pain, n (%)	232 (12.9)
	Moderate Back Pain, n (%)	315 (17.6)
	Severe Back Pain, n (%)	164 (9.1)
Back Injury	No Back Injury, n (%)	1752 (97.7)
	Has had a Back Injury, n (%)	41 (2.3)
Spine or Facet OA	No Spine or Facet OA, n (%)	325 (18.0)
	Facet OA Only, n (%)	408 (22.8)
	SOA Only, n (%)	236 (13.2)
	Both Spine and Facet OA, n (%)	824 (46.0)
Knee OA	No Knee OA, n (%)	1087 (60.6)
	Has Knee OA, n (%)	706 (39.4)
Hip OA	No Hip OA, n (%)	1205 (67.2)
	Has Hip OA, n (%)	588 (32.8)
Hand OA	No Hand OA, n (%)	1244 (69.4)
	Has Hand OA, n (%)	549 (30.6)

[§]Restricted to those with non-missing spine-facet OA and covariates

Table 2.

Characteristics by categories of osteoarthritis (OA) in the lumbar spine (n=1793)[§]

Baseline Characteristic	Level	No FOA or SOA	FOA only	SOA Only	FOA and SOA	P-value
		n=325 (18.0%)	**n=408 (22.8%)**	**n=236 (13.2%)**	**n=824 (46.0%)**	
Age	Age, Years, Mean (SD)	59.6 (9.1)	65.2 (9.6)	64.2 (9.5)	69.8 (9.2)	<.0001
	Age <55, n (%)	122 (45.5)	63 (23.5)	38 (14.2)	45 (16.8)	<.0001
	Age ≥55-<65, n (%)	122 (20.3)	155 (25.8)	92 (15.3)	232 (38.6)	
	Age ≥65-<75, n (%)	58 (10.5)	123 (22.2)	74 (13.4)	298 (53.9)	
	Age ≥75, n (%)	23 (6.2)	67 (18.1)	32 (8.6)	249 (67.1)	
Baseline	Baseline @ T1, n (%)	227 (27.8)	200 (24.4)	117 (14.3)	274 (33.5)	<.0001
	Baseline @ T2, n (%)	98 (10.1)	208 (21.3)	119 (12.2)	550 (56.4)	
BMI	Mean BMI, Mean (SD)	29.5 (5.8)	31.2 (6.4)	30.2 (6.7)	31.0 (6.1)	0.0002
	BMI <25, n (%)	74 (24.4)	67 (22.1)	46 (15.2)	116 (38.3)	0.0019
	BMI 25-<30, n (%)	120 (19.5)	126 (20.5)	82 (13.3)	288 (46.8)	
	BMI ≥30, n (%)	131 (15.0)	215 (24.6)	108 (12.4)	420 (48.1)	
	Missing, n (%)	29.5 (5.8)	31.2 (6.4)	30.2 (6.7)	31.0 (6.1)	0.0002
Gender	Men, n (%)	147 (22.7)	119 (18.3)	91 (14.0)	292 (45.0)	<.0001
	Women, n (%)	178 (15.6)	289 (25.3)	145 (12.7)	532 (46.5)	
Race	African American, n (%)	138 (24.2)	133 (23.3)	93 (16.3)	206 (36.1)	<.0001
	White, n (%)	187 (15.3)	275 (22.5)	143 (11.7)	618 (50.5)	
Back Pain	No Back Pain, n (%)	200 (18.5)	259 (23.9)	133 (12.3)	490 (45.3)	0.2951
	Has Back Pain, n (%)	125 (17.6)	149 (21.0)	103 (14.5)	334 (47.0)	
Pain Severity	No Back Pain, n (%)	200 (18.5)	259 (23.9)	133 (12.3)	490 (45.3)	0.4358
	Mild Back Pain, n (%)	38 (16.4)	40 (17.2)	36 (15.5)	118 (50.9)	
	Moderate Back Pain, n (%)	53 (16.8)	72 (22.9)	47 (14.9)	143 (45.4)	
	Severe Back Pain, n (%)	34 (20.7)	37 (22.6)	20 (12.2)	73 (44.5)	
Back Injury	No Back Injury, n (%)	319 (18.2)	400 (22.8)	225 (12.8)	808 (46.1)	0.0762
	Has had a Back Injury, n (%)	6 (14.6)	8 (19.5)	11 (26.8)	16 (39.0)	
Knee OA	No Knee OA, n (%)	263 (24.2)	259 (23.8)	154 (14.2)	411 (37.8)	<.0001
	Has Knee OA, n (%)	62 (8.8)	149 (21.1)	82 (11.6)	413 (58.5)	
Hip OA	No Hip OA, n (%)	235 (19.5)	290 (24.1)	170 (14.1)	510 (42.3)	0.0002
	Has Hip OA, n (%)	90 (15.3)	118 (20.1)	66 (11.2)	314 (53.4)	
Hand OA	No Hand OA, n (%)	278 (22.3)	294 (23.6)	180 (14.5)	492 (39.5)	<.0001
	Has Hand OA, n (%)	47 (8.6)	114 (20.8)	56 (10.2)	332 (60.5)	

§Restricted to those with non-missing spine-facet OA and covariates; FOA=facet joint OA only; SOA=spine OA only; SOA and FOA=spine OA and facet joint OA

Table 3.

Unadjusted and Adjusted[‡] odds ratios and 95% confidence intervals for the association between explanatory variables and the categories of osteoarthritis (OA) in the lumbar spine (n=1793)

	FOA only n=408 (22.8%)		SOA only n=236 (13.2%)		Both FOA and SOA n=824 (46.0%)	
	Unadjusted OR (95% CI)	**Adjusted OR (95% CI)**	**Unadjusted OR (95% CI)**	**Adjusted OR (95% CI)**	**Unadjusted OR (95% CI)**	**Adjusted OR (95% CI)**
Age <55	Ref	Ref	Ref	Ref	Ref	Ref
Age ≥55-<65	**2.46 (1.67–3.62)**	**1.84 (1.19–2.84)**	**2.42 (1.54–3.81)**	**1.83 (1.11–3.03)**	**5.16 (3.43–7.74)**	**2.99 (1.90–4.72)**
Age ≥65-<75	**4.11 (2.66–6.35)**	**3.01 (1.81–4.99)**	**4.10 (2.48–6.76)**	**3.01 (1.69–5.35)**	**13.9 (8.95–21.7)**	**7.13 (4.29–11.8)**
Age ≥75	**5.64 (3.21–9.90)**	**4.07 (2.09–7.90)**	**4.47 (2.34–8.54)**	**3.10 (1.45–6.62)**	**29.4 (17.0–50.7)**	**13.3 (6.98–25.2)**
BMI <30	Ref	Ref	Ref	Ref	Ref	Ref
BMI ≥30	**1.65 (1.23–2.22)**	**1.76 (1.28–2.42)**	1.25 (0.89–1.75)	1.20 (0.84–1.72)	**1.54 (1.19–2.00)**	**1.85 (1.37–2.51)**
White	Ref	Ref	Ref	Ref	Ref	Ref
African American	**0.66 (0.48–0.89)**	**0.68 (0.49–0.95)**	0.88 (0.63–1.24)	0.97 (0.67–1.39)	**0.45 (0.34–0.59)**	**0.51 (0.37–0.70)**
Men	Ref	Ref	Ref	Ref	Ref	Ref
Women	**2.01 (1.48–2.72)**	**1.71 (1.24–2.36)**	1.32 (0.94–1.85)	1.16 (0.81–1.65)	**1.50 (1.16–1.95)**	1.19 (0.88–1.60)
No Back Pain	Ref	Ref	Ref	Ref	Ref	Ref
Has Back Pain	0.92 (0.68–1.24)	1.00 (0.73–1.39)	1.24 (0.88–1.74)	1.38 (0.96–1.99)	1.09 (0.84–1.42)	**1.35 (1.00–1.84)**
No Back Pain	Ref	Ref	Ref	Ref	Ref	Ref
Mild Back Pain	0.81 (0.50–1.31)	0.86 (0.52–1.42)	1.42 (0.86–2.36)	1.50 (0.89–2.55)	1.27 (0.85–1.89)	1.40 (0.89–2.20)
Moderate/Severe Back Pain	0.97 (0.69–1.35)	1.08 (0.75–1.55)	1.16 (0.79–1.70)	1.32 (0.88–1.99)	1.01 (0.75–1.37)	1.33 (0.94–1.87)
No Back Pain	Ref	Ref	Ref	Ref	Ref	Ref
Mild Back Pain	0.81 (0.50–1.31)	0.86 (0.52–1.42)	1.42 (0.86–2.36)	1.50 (0.89–2.54)	1.27 (0.85–1.89)	1.40 (0.89–2.20)
Moderate Back Pain	1.05 (0.70–1.56)	1.12 (0.73–1.72)	1.33 (0.85–2.09)	1.50 (0.94–2.40)	1.10 (0.77–1.57)	1.32 (0.88–1.98)
Severe Back Pain	0.84 (0.51–1.39)	1.00 (0.59–1.70)	0.88 (0.49–1.60)	1.02 (0.54–1.90)	0.88 (0.56–1.36)	1.36 (0.82–2.24)
No Back Injury	Ref	Ref	Ref	Ref	Ref	Ref
Has had a Back Injury	1.06 (0.37–3.10)	1.50 (0.49–4.59)	2.60 (0.95–7.13)	**3.11 (1.09–8.91)**	1.05 (0.41–2.71)	1.50 (0.53–4.27)
No Knee OA	Ref	Ref	Ref	Ref	Ref	Ref

	FOA only n=408 (22.8%)		SOA only n=236 (13.2%)		Both FOA and SOA n=824 (46.0%)	
	Unadjusted OR (95% CI)	**Adjusted OR (95% CI)**	**Unadjusted OR (95% CI)**	**Adjusted OR (95% CI)**	**Unadjusted OR (95% CI)**	**Adjusted OR (95% CI)**
Has Knee OA	**2.44 (1.73–3.44)**	**1.78 (1.23–2.57)**	**2.26 (1.54–3.32)**	**1.79 (1.19–2.70)**	**4.26 (3.13–5.80)**	**2.50 (1.78–3.52)**
No Hip OA	Ref	Ref	Ref	Ref	Ref	Ref
Has Hip OA	1.06 (0.77–1.47)	**0.69 (0.48–0.98)**	1.01 (0.70–1.47)	0.67 (0.45–1.01)	**1.61 (1.21–2.13)**	0.74 (0.53–1.03)
No Hand OA	Ref	Ref	Ref	Ref	Ref	Ref
Has Hand OA	**2.29 (1.57–3.34)**	1.16 (0.76–1.77)	**1.84 (1.20–2.83)**	1.12 (0.69–1.81)	**3.99 (2.84–5.60)**	1.37 (0.92–2.03)

[ǂ]All explanatory variables simultaneously considered in model; FOA=facet joint OA only; SOA=spine OA only; SOA and FOA=spine OA and facet joint OA; Referent group = neither FOA nor SOA n=325 (18.0%)